A STUDY OF MUSCÆ, WITH SUGGESTIONS AS TO THE PHYSIOLOGY OF INTRAOCULAR NUTRITION, THE ETIOLOGY OF CATARACT, GLAUCOMA, ETC.

BY

GEORGE M. GOULD, A.M., M.D.,
OF PHILADELPHIA

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INTRODUCTORY. Subjective visual sensations have not been studied by scientific methods to that extent that their importance warrants. That these phenomena are very common (occurring doubtless to every person), of very great variety, of physiologic significance, and that they are transitory symptoms of many diseases, and unexceptional or continuous symptoms of others—these facts have long been known, but their import has hardly been realized. The recent admirable study of one variety, scotoma scintillans, by Gowers,[1] is, so far as I know, almost the first, certainly the first scientific attempt to classify one genus of these phenomena. Before reading Dr. Gowers' lecture I had carried out the experiments (if they may be so dignified) and made the notes to be described hereafter. Encouraged by the illustrious example, I am moved to set forth a few observations that may be suggestive and helpful to others in their better studies.

[1] Lancet, Nos. 3747 and 3748. The Bowman Lecture: On Subjective Visual Sensations.

CLASSIFICATION. Until more accurate observations have been made, any attempted classification and theory of the phenomena in question will be inexact and incomplete. So soon as possible some sort of grouping and ordering should be made, however tentative it may be. But any such classification at once presupposes a theory or doctrine assumed concerning them, and this assumption is, of course, met by the evident fact that, scientifically, we know too little concerning them to permit of any but a loose and conjectural theory. In a rough way, however, with some doubt and qualification, we may say that all the phenomena in question arise or reside in organs more or less localizable—*i. e.*, they are either:

1. *Peripheral,* originating in the globe of the eye (including the optic nerve), or in adjacent and related organs, as, *e. g.*, muscæ, pressure-phosphenes, etc.
2. *Central,* originating in the cerebral centers where optic-nerve impulses are transformed into visual sensations, as, *e. g.*, scotoma scintillans, phosphenes from occipital traumatism, etc.
3. *Combinations of the two.*
   a. Peripherocentral, primarily or preponderatingly peripheral.
   b. Centroperipheral, primarily or preponderatingly central.

In a strict and physiologic analysis it may be true that every visual sensation requires the synchronous cooperation of both the peripheral and the

central mechanisms. In those most manifestly central the mind imperatively locates a corresponding object outside of the body, and even a corneal opacity requires the central mechanism to become subjectively manifest. And yet the classification has a certain validity readily understood.

NOMENCLATURE. This also is in a state of chaos. It would be easy to devise a systematic nomenclature. I have, indeed, busied my fancy with one—and present it with little confidence, because such Minerva-born systems are generally stillborn. The world loves its old foolish names of things, and with most people habit is fate. It would seem a clarifying and labor-saving device to designate somewhat as follows:

SUBJECTIVE VISUAL SENSATIONS.

PHOSES: Light-sensations of whatever kind or color, of a positive nature.

APHOSES: Absences or interruptions of light-sensations, scotomata, shadows, whether formless or of more or less indeterminate form.

PERIPHEROPHOSES: Phoses originating in the peripheral ocular mechanism.

CENTROPHOSES: Phoses originating in the central ocular mechanism.

PERIPHERAPHOSES: Aphoses originating in the peripheral ocular mechanism.

CENTRAPHOSES: Aphoses originating in the central ocular mechanism.

CHROMOPHOSES: Colored phoses—and, if desirable, subvarieties, to wit:

a. *Erythrophoses*, Red Phoses.

b. Chrysophoses, Orange Phoses.
c. Xanthophoses, Yellow Phoses.
d. Chlorophoses, Green Phoses.
e. Cyanophoses, Blue Phoses.
f. Indikophoses, Indigo Phoses.
g. Ionophoses, Violet Phoses.

Without driving the name-making instinct to name-mania, the modifications and qualifications of all subjective visual sensations may be adapted to the terms given, as, *e. g.*, stellate, fiery, darting, stationary, annular, beaded, fortification phoses, etc. The word *spectra* should not be used here, as it has other definite and sufficient uses.[1]

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[1] A Personal Observation as to "Fortification"-Chromophoses. Dr. Gowers has wisely suggested the collation of personal experiences, and although my object in this writing is to express some thoughts as to one variety of "peripherapheses," commonly called *muscæ volitantes*, which should be conveniently contracted to *muscæ*, I shall take occasion to say parenthetically that I have been subject all my life to attacks of fortification-chromophoses, or scotoma scintillans. These seem to have no definite periodicity. Sometimes I may go a year with none, and sometimes I may have several attacks in as many weeks. I do not remember to have had two attacks on the same day. I can trace them to no cause either in the general health, or in special conditions, of study, etc. I think being in bright sunlight has sometimes been an immediate or cooperating factor in bringing on an attack. More often I have noticed that after walking rapidly in sunlight the sudden entrance into a dark hall or room would produce what might be called a pseudo-attack—an aura, or intimations of the same, dim scintillations, indistinct chromophoses, but lasting only for a few seconds or a minute.

The genuine attack begins with the dimmest fluttering, as if of some indeterminate thing at the periphery of the field of vision, usually on the right side, a little north of east, let me say, or sometimes due northeast. This fluttering gradually approaches the center of the field, but never reaches it, and as it approaches

PERIPHERAPHOSES may, of course, be due to opacities of the cornea, aqueous humor, lens, vitre-

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it becomes both more distinct in form and more definite in chromatization. It takes from five to ten minutes before the acme of the attack is reached, and as long for the subsidence, which last, as to color, location, etc., is the exact reverse of that of the ascending process. At the period of greatest intensity the chromatization is not definite; *i. e.*, I cannot clearly distinguish definite colors; this may be due to the rapidity of the fluttering or scintillating movements, which I estimate as about six or eight a second. This vibratory or fluttering movement is about uniformly rapid from the beginning to the end of the attack, and is about the same in all attacks.

The predominating colors, perhaps, are, in order of prominence, yellowish-white, reddish-yellow, and a dark neutral-tinted or aphosic line that forms the most clearly distinguished central part of the "fortification"-figure. But the angles of my fortifications are never sharply acute; they are usually about right-angled, or slightly obtuse. Sometimes in their approximation to the center of the field of vision the widening fluttering lines will show a distinct curvature about it, but neither the straight nor the curved series of angular chromophoses usually pass beyond the hemianopic vertical line into the left hemisphere. There are no sequelæ whatever, no migraine, no headache, or other uncomfortable symptom.

During the paroxysm, although the chromophoses do not seem to attack the macular vision directly, I somehow find it impossible to continue reading. I have a difficulty in determining or describing the reason for this. By an effort of will I can see the printed words, at least temporarlly, but they become blurred or in some way indistinct, and it becomes extremely difficult, though not painful, hardly even uncomfortable, to continue. The waving and quivering of that tormenting flag, so very close to the eye, is unendurable, and I lie down or walk about until it all passes away. Position of the body, or brisk exercise—nothing seems to influence in the least the ordered march of events. The process seems to appear when it will, without discoverable cause, and carries out its kaleidoscopic program without regard to any physiologic influence.

I may also allude to a variety of phoses that I have observed a

ous, or retina. The most constant, commonly observed, and often bothersome of these is the class called muscæ volitantes (better, simply muscæ), or, perhaps, muscal peripheraphoses.

Objective Conditions for Studying Muscal Peripheraphoses. In the summer of 1895 I lived upon a mountain 4000 feet above sea-level. In looking at the panorama of mountains and sky stretched before me I found that my never-absent *mouches volantes* were often outlined with surprising clearness, and were far more numerous than I had supposed. I found that the background of a sun-illumined bank of clouds brought them out with unsuspected sharpness. I have spent many hours in studying them. The dull white, horizonless, cloudy, misty, or smoky sky, with no definite object to fix the attention, below, around, or above, is, therefore, the best objective condition for the study proposed.

Subjective Conditions. The principal of these is good health (as that is essential to the highest

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thousand times, but that I have not seen described. In looking at a uniformly well-lighted blue sky I can, by careful and continuous indeterminate observation or interspace-fixation, bring to view numberless pin-point phoses, not very sharply and definitely outlined, but still clearly seen, appearing and disappearing with almost lightning-like rapidity, and quivering or darting throughout the central visual field. I attribute these to the commotion set up by the diffused strong light-stimulus in the pigmentary receiving-substance of the retina, these individual dot-phoses or pepheropheroses being the idiopathic response of particular retinal nerve-ends or elements to the general stimulus. These definite phoses are preceded by a period of indeterminate quivering of the air, as it were, or of something so near nothing that it is beyond recognition or description.

retinal sensitiveness), patience, dexterity of ocular movements, good visual acuteness (my own is 20/10), and experience in catching infinitesimally slight visual sensations, and especially those intermediated by peripheral portions of the retina. The less direct sunshine there is the wider the pupils, and hence the larger the "field of operation," although the clouds should be bright with diffused sunlight. A large measure of success is doubtless dependent upon long education of the extramacular portions of the retina, and the ability to study objects focused there. But I shall describe a most valuable means of largely obviating the necessity of this peripheral-field study, and one also that has, I think, considerable significance in other directions. This method is one that for want of a better name I have called—

Ocular Ballottement. It is almost needless to explain the principle of physics underlying the experiment of ballottement as practised by obstetricians. If a body of slightly greater specific gravity than the liquid in which it is submerged is, by a fillip, thrown to the surface or upward, it will slowly settle back again to the bottom. In cases of chronic choroiditis shreds of exudate floating in a liquid vitreous can, by a little fillip of the eye by the patient (first upward and then suddenly halting at horizontal fixation), be seen with the ophthalmic mirror descending to the lower part of the eye like snowflakes in the night as seen from a window. The rate of downward movement will, of course, depend upon the relative densities of the liquid and the opacities.

From time immemorial muscæ have been described as shadows of retinal vessels and vitreous cells directly *in front of the retina* (*not* directly in front of the *vitreous*), located at one side of the fixation-point, so that they continually elude direct fixation by a will-o'-the-wisp movement ever further and further as one seeks to transfix them with the optic axis. A few experiments with ocular ballottement will show one that these are not fixed particles, but that they are bodies floating in a liquid medium, and thrown by motions of the eyeball upward or about, to settle, if permitted, at the most dependent portion of the eye. This can be demonstrated, while sitting in the usual position, by a rapid whirl of the eyeballs, suddenly again resting at horizontal fixation. The swirl of the muscal bodies will now be seen to follow the previous circular motion of the liquid, curving as they fall. One may then lie down on the right side with the axis of the body and head horizontal: the particles now fall, after ocular ballottement, toward the right temple or the ground. By lying on the left side the same facts, *mutatis mutandis*, are shown. By a very sudden and energetic jerk of the eyes downward, arresting the movement very sharply and suddenly, one may get a positive rebound from the bottom of the chamber up to the line of visual perception. But the most convincing and instructive experiment is to lie flat on one's back, without pillow and with the chin slightly elevated; by dexterity both in movements of the head and of the eyes, one is then able to bring any one of these muscæ into the center of the field, and poise it there, as a juggler adjusts a whirling

plate above his head. Thus to bring any one of many muscæ haunting the periphery into the line of macular vision, and to hold it steadily there while gazing at it, will require considerable experimentation with the ocular fillip, and with delicate poising or adjustment of the head. By alternately closing one eye and then the other one may observe in which eye a group of muscæ is located.[1]

Action of Accommodation upon Muscæ. If while holding an aphose thus in unstable but continuous fixation, a sheet of gray paper is interposed within about twelve inches of the eyes, at once what was a very indeterminate, formless, or dimly seen shadow is minimized in size, but correspondingly definitized in outline and characteristics according to well-understood optic laws. At the same time the fact will be noted that when deftly done the act of accommodation does not *per se* squeeze, or deport the object aside. I infer from this that wherever the liquid chamber is located the bulging of the crystalline lens does not greatly displace the particles of the liquid and of the contained muscægenetic particles. As I shall try to show, this liquid chamber (let us for the time name it the aqueovitreous chamber) is, I believe, located immediately behind the lens, and I at first thought that the fact that the particles do not move by the act of accommodation argued against the theory of this location of the chamber, because it would seem that the backward

[1] I have been able to observe muscæ through the closed lids, the eyes being directed toward brilliant light. I have also studied them face downward, overlooking a neutral-tinted illumined porch-floor, but the experiments brought out no significant facts.

bulging of the lens would somewhat squeeze aside the particles in the base of the cup. I conclude, however, that the chamber is located as described, and that the non-movement of the particles in accommodation is due to the fact that the chamber is *full* of liquid and under pressure as much, of course, as all intraocular humors, and that the slight backward thrust or compression would not, therefore, displace the particles in the central portion, or that pierced by the visual axis. Besides this, the bulging of the circumjacent ciliary muscle would equal and neutralize any tendency of the lens-movement to displace them.

The Location of the Aqueovitreous Chamber. The motion of the muscægenetic particles after the fillip of ocular ballottement determines the location of the aqueovitreous chamber. Objectively in choroiditis we can see the opacities descend after the upward glance of the patient's eye, or rebound from the bottom of the chamber when a sharp downward sweep is suddenly arrested. Subjectively we may observe the same phenomenon in the case of physiologic peripheraphoses or muscæ. But in the latter instance we can use the slightest fillip, a mere hint of short upward-glancing, and observe the resultant upward jerk of the particles, to be followed, of course, by their slow fall if the gaze is then kept steady and unmoved. Sometimes the particle is situated laterally in the field, and the motion of the eye to see it continuously displaces it further laterally, but the usually downward tendency can be shown by steadily holding the eye quiet. It certainly falls by gravity, out of

reach of sight, in the dark parts of the chamber. I have rarely noticed any appearance disproving that the particles are not freely mobile, except that the larger or more swollen strands, and especially the tangled masses of the same, are often more difficult to manage, seem to adhere to the walls of the chamber, etc. These facts necessarily locate the chamber in front of the vertical equator of the eye. If the chamber were posterior to the equator, an upward glance would throw the particles downward. (This fact may serve as an aid in localizing the position of pathologic vitreous opacities.) From readily recognized reasons, needless to recapitulate, the location of the aqueovitreous chamber containing the muscægenetic particles in suspension must be between the vitreous body posteriorly and the lens, its ligament, and the ciliary body, anteriorly. It may be said if such a "chamber" existed in this situation anatomists would long ago have demonstrated its existence, but in reply it need only be said that the mere fact of opening the globe destroys the space, and the extremely thin film of liquid in the unwalled space disappears unnoticed. Attention to it will, I am sure, result in methods of demonstrating its existence with the naked eye or the microscope.

Character of the Muscægenetic Particles. In my own case the most conspicuous of these particles are what I have called cellular; *i. e.*, aphosic globes or ring-like appearances, sometimes nucleated (with a darker center), the nucleus surrounded by a space of lighter gray between it and the darker wall. With suspended accommodation these cells,

as shown against a cloud, seem to subtend an angle of perhaps one or two minutes, and all outlines are softened or blurred. With the accommodation active, these cells, with their (four-fifths or nine-tenths?) reduction in size, gain definiteness of contour and structure, and what seemed before as discrete cells are usually found to be arranged in chains or necklaces, the cells often marking an angle or curving-point from which extends an indistinct and homogeneous band or cord running off to other cells.[1] Almost constantly there are in the field also continuous strings of cells, the intercellular cord seemingly non-existent, and lying in contact with one another. I have counted as many as twenty-five or thirty, or more, "beads" or cells in a single string. Sometimes the strings stretch out almost in a straight line; at other times they are coiled and tangled in a confused mass. They change in appearance every second, as they are moved by the currents of the liquid and motions of the eyeballs. Under the microscope or in illustrations of histologic teased tumor or other tissue, I have seen structures almost exactly like those of certain muscæ vary in their relative density, some falling more rapidly than others. A few seem almost of the same specific gravity as the liquid, and fewer still are lighter. In the same string the beads are almost uniform in size, but are of different sizes in different strands, some being twice the size of others. The larger are usually of indefinite outline and more filmy in ap-

[1] It was this, I think, that v. Zehender saw in his experiment, and not a reflex of the optic nerve.

pearance, seemingly more swollen and nearer disintegration.

The second class includes mere indeterminate and formless, cloudy or filmy shadows, or shreds of all sizes.

The third class requires for observation extraordinary acuity of vision, long experience and education in ocular ballottement, as well as peculiar and almost indescribable conditions of light. Through a window-slit I have sometimes observed them with remarkable distinctness, upon awakening in the morning, with the retina, of course, in a rested and acutely sensitive condition. They seem like dust or fine rain, settling downward much more slowly after the upward ocular fillip.

As nearly, therefore, as I can observe and classify them, my muscæ are:

1. Cells, or cell-strands, sometimes with intercellular cords, sometimes in juxtaposition, like a close necklace of beads.

2. Formless filmy or cloudy masses.

3. Fine particles, in appearance like soft, fine rain.

I have thus described these aphoses as they appear in a sitting posture, but by lying flat upon one's back and poising them before the macula the second class is soon proved to consist usually of the chains or necklaces that seemed formless only because they were seen by the peripheral portions of the retina, or because matted and massed in a tangle. Such a seemingly formless mass, poised before the macula may even be unraveled or disentangled by numerous ocular ballottements, until all the inter-

laced and matted strings are washed apart from each other.

Experience and careful observation have convinced me that the quantity of muscægenetic material is vastly greater than has been suspected. Under exceptional circumstances of light, etc., I have certainly observed as many as twenty or thirty separate strands or masses in the field at one time. In my own case I am in perfect health, and the seemingly excessive amount of this material cannot, therefore, be called pathologic. I believe that, in varying degrees, the material is present in every person, requiring only a number of necessary conditions and sufficient attention to make it manifest. While reading, of course, the lateral motions of the eyes do not make this vastly larger part of particles surge upward into the field of view, either to obscure vision or to become noticeable, and when walking they are not observable by anyone. If the motion of the eyes in reading were up and down, they would certainly be more troublesome. While still physiologic the amount of matter is never so great as distinctly to cloud vision, except in microscopic work or in such climatic surroundings as are fitted to bring it before the attention. Then it must not be forgotten that only as it rises into or descends through the small space lighted by the pupil is it possible for the retina to note its existence. There is doubtless, all about, a cavity or peripheral portion of the aqueo-vitreous chamber large in extent as compared with the area illuminated through the pupil, because by ballottement the muscæ fall out of sight and rise again out of an unseen space. Moreover, the periph-

eral muscæ at one time in the field never occupy what I should judge to be over 40° or 50° of the extramacular field. Even within this space, however, the trained and sensitive retina directed to the zenith perceives a vastly larger amount of muscæ-genetic material than would have been believed possible. It may also be noted that out of doors, or in well-lit rooms, the pupil is contracted, whilst in mydriasis the conditions of observation and study are destroyed.

There is a toy that crudely but strikingly illustrates the manifest fact. It is a hollow glass globe, filled with fluid, at the base of which are illustrated a house, people, etc. The fluid contains a sort of sand, of slightly heavier specific gravity than the liquid. By shaking the white sand a mimic "snow-storm" is produced as the grains settle to the bottom. If this globe were surrounded with black paper, except at one point, leaving an imitation pupil, and at the opposite end a hole to look into, and if into the liquid were thrown diaphanous or translucent shreds of teased and broken-down tissue in all states of degeneration, by manipulations, or "ballottements," one would have all the essential phenomena of the eye described, providing the size and refraction of the globe were such as to create the verisimilitude. The "chamber" of the eye, instead of being round and roomy as in the toy, is but a thin vertical space, between the vitreous and the structures in front of it, possibly thinner in the center, and deepening at the periphery adjacent to the ligament of the lens. Never have I seen a strand of cells as if "end-on," and rarely more

than one or two behind another. In the region pierced by the visual axis, the chamber is therefore not much deeper than two or three times the thickness of the lining membrane of a vitreous cell.

The Function of the Aqueovitreous Chamber and its Contents. What an organ does, that is its function—and its functions, as those of most structures, are often multiple and made to serve multiple purposes. It is the fault of physicians that, having discovered one use of an organ, or one factor in the etiology of disease, they rest content and do not learn that, generally, disease has more than one, often several causes. In physiology, *e.g.*, the object of winking has always been given as the cleansing and the keeping of the cornea uniformly moist. Doubtless a result as absolutely important is that of giving the retina temporary rests of darkness, whereby the extremely complex and unstable sensitiveness (visual purple or what not) is re-established. One may doubt if there is a more difficult and enormously delicate labor carried on by the animal economy than that of first making and then keeping the retina in that receptively delicate condition necessary for response to the inconceivably and infinitesimally slight stimulus of light. Muscæ may be considered as one such aid in securing an instant's rest, in temporarily shading, and continuously replacing light by shade, upon which the retinal equilibrium depends. Thousands of times every day these shadowy particles, ever variant in size and qualities, glide unnoticed in front of the macular region, and together with winking give it

an instant's reduction or relaxation from the strain of visual response. But other and more important functions are suggested.

1. The existence of such a chamber serves to unravel the mystery of, at least, one aspect of vitreous nutrition, disconnected, as the vitreous body so largely is, from the usual means of excreting waste products. The hyaloid canal aids lymphatic excretion through the optic nerve, but both it and the hyaloid membrane are open in front and permit drainage into the aqueovitreous chamber there located. Into this space are extruded the products of tissue-metamorphosis, the collapsed worn-out cells, shreds of tissue—in a word, the débris of vitreous katabolic change.

2. The liquid may have a useful purpose as a lubricant and buffer in the ceaseless changes and frictions of the mechanism of accommodation, in equalizing and distributing pressure, etc. If the vitreous were pressed close against the filtering membrane, the passage of any liquid through the septum into the aqueous chamber would be greatly impeded.

3. Possibly more important still, the chamber may have a most useful and necessary function as a "settling-basin," wherein the undissolved products of vitreous excretion are shoved, and thus gotten out of the way of direct vision so far as relates to *fixed* and *continuous* opacifications. What a nuisance it would be to have a single musca-cell permanently before the macula!

4. As is well known, the liquid of the first exhaustion of the aqueous chamber is non-albuminous

and non-coagulable. Upon the reformation of the aqueous a second tapping is found to yield an albuminous and coagulable fluid. The significance of this fact in wounds of the cornea is evident. May the aqueovitreous chamber not aid in this function and serve as a storehouse or reserve stock for refilling the aqueous chamber, and in case of traumatism, with a readily coagulable fluid?

5. Doubtless by far the most important use, however, is that of attrition, dissolution, breaking-down, and liquefaction of the débris of vitreous excretion, so that this may pass into the absorbent channels and thus finally be disposed of. The process to which these particles are subjected by the ceaseless motions of the eyes is a genuine churning-process, the narrowness of the space in which they are confined rendering it all the more effective in the uninterrupted friction of the particles against each other and against the boundaries of the chamber. It may also be possible that the liquid may have some true digestive power to hasten the process of liquefaction. Active out-of-door life begets healthier eyes than indoor life, and to some extent this may be due to the more effective nutrition resulting from better local conditions of attrition, etc., in the "churning-process," resulting from bodily and ocular motility.

6. The translucent muscægenetic particles floating in the chamber may afford the incidental shading of all parts of the macular region to which allusion has been made, and thus serve to give slight relief from the constant glare of light that, as all know, is so harmful to retinal sensitiveness. In this respect

they serve to supplement the act of winking, as every wink slightly stirs the sediment of the liquid, just as to a greater extent does every contraction of the extra-ocular muscles, incessantly active during waking.

The query arises and may be worth noting parenthetically, whether the products of this dissolution-process or digestion may not in some way be utilized by the lens as nutritive material. So large an amount of organic matter, half or wholly digested and lying in constant contact with the capsules and ligaments of that organ, the only supply of nutriment of which is drawn from this cavity, must certainly exercise an important influence upon its nutritional condition and repair. But this aspect leads me to remark upon a possibly pathologic significance growing out of the ensemble of facts and inferences that have been set forth.

The two most common, most mysterious, most serious, and, to treatment, most intractable, of all diseases of the eye are cataract and glaucoma. The etiology of each is as obscure as the treatment is unsatisfactory. That the best minds of a special class of brilliantly intellectual medical men have failed so far to bring solutions to these questions makes me shrink from any but the most modest suggestion of a not improbable explanation.

A Possible Factor in the Etiology of Cataract. Whether or not the lens and its capsules, for purposes of nutrition, make any physiologic use of the dissolved and digested organic matter bathing the posterior capsule and ligament, it does not seem otherwise than reasonable that with path-

ologic conditions of this liquid there may ensue pathologic conditions of the lens and disturbances of its nutrition. Careful consideration of the influences and circumstances at work, of the transcendent delicacy of the processes, and of the instability of the equilibrium or balance between secretion and excretion of the "closed system" of the eye—all these and other things that might be enumerated make it plain that pathologic states of the liquid of the aqueovitreous chamber may readily arise. When one reflects upon the unsuspectedly large amount of normal undissolved muscægenetic material or débris of this chamber, it is readily seen that a slight increase in the amount above the normal would readily change physiology into pathology. Under such circumstances digestion would quickly become dyspepsia—and especially so in the already deteriorated nutritional processes of the aged, of the weak, etc. Then, also, it should be remembered that the slightest decrease of the amount of the diluent, the secretion filling the chamber, would have the same effect. Let one also realize well the necessarily small capacity of the chamber, the general microscopic plan of the space, the parts and processes, and it will become plain that the narrow line separating physiology and pathology is most easily passed. Senile cataract does not arise until the late period of life when nutritional disturbances are at work in the entire organism and the lessened vital forces are more and more incapable of repairing the ravages of tissue-waste and injury. I have myself no doubt that long-continued uncorrected eye-strain is a potent factor in the pro-

duction of senile cataract, but that it acts alone, without other contributing agencies, is hardly supposable. With the growing inelasticity of the lens—presbyopia—(may this inelasticity itself not depend upon local (muscal?) as well as systemic denutrition?) the presbyopic ciliary muscle does not cease to work—indeed, its labors are perhaps all the more straining and irritating. Uncorrected astigmatism in presbyopes thus serves to induce pathologic conditions of the ligament and capsule of the lens (the filtering membrane), with irritation and denutrition. I would only hazard the suggestion that a morbid condition of the fluid bathing the parts, an excess of undigested muscæ, a slight upsetting of the balance between secretion and excretion, may cooperate with ametropia and systemic denutrition to produce the local lenticular denutrition or morbidity called cataract.[1]

The Etiology of Glaucoma. The obliteration of the filtration-angle is beyond question the immediate or secondary cause of glaucoma, but what is the *causa causæ*—the cause of the cause? Why the increased pressure from behind? To this question no answer has been given. Closure of the filtration-angle or of the canal of Schlemm, and excessive secretion in the vitreous chamber—deficient excretion in front, and over-production behind—have seemed the sole alternatives. To these I would

[1] Certain forms of anterior choroiditis, cyclitis, "posterior capsulitis," etc., may possibly be caused or complicated by such conditions. Bacteria introduced through traumatism or other means would find here a culture-medium well adapted for their multiplication.

add a third, the choking or clogging of the sieve, the membranous septum of the lens-ligament, by the débris-sediment, or solution of the aqueo-vitreous chamber. Excreted organic matter, softened, partially liquefied, or partially digested, granular, or viscid, of all possible substances, would be most perfectly adapted to choke up the pores and the intercellular spaces of a membranous diaphragm. This is so outside of the body in the osmotic processes of dead membranes, and it cannot fail to occur in the living membrane.

It may be urged that such a choking of the septum would at best only be likely at the bottom of the chamber, where the sedimental particles naturally settle. The objection ignores three facts:

1. It is not at all to be taken for granted that only it is the visible, the heavier, or the larger particles that may choke the membrane the most. It may possibly be the lighter and finer, the more dissolved varieties that are not seen at all, or that are rarely seen; or, still more probable, the liquid itself, made over-viscid by too much dissolved material.

2. We do not know but that the top of the chamber may be equally or even more charged with non-liquefied, unabsorbed material of lighter specific gravity than the liquid. This would seem very plausible. Such particles would not often produce muscæ, because, to reason from analogy with objective solutions, such floating matter would, by the motions of the eye, not be submerged, and thus be brought into the area to render them visible. That some muscægenetic particles are lighter than the liquid,

and do inhabit the upper part of the chamber, I have (rarely) proved.

3. Most significant still is the answer that we forget that one-third of our lives is spent in sleep and in lying down. Those who sleep on their sides would expose one of the lateral (when prone, the lowermost) portions of the chamber to impacts of the sedimentation-process, and at a time when immobility of the eyes would permit the injury (if it exist) from sedimentation and choking to be most active. Very few persons lie habitually and continuously all night with the head in one position, so that different parts of the filtering septum are successively exposed for hours to the risk of being covered with an excess of sediment. It is said that in sleep the eyes are turned upward; *i. e.*, as if by contraction of the superior rectus. If so, is it a device of the organism to permit the sediment to drop away from the septum, and at the posterior or flat aspect of the chamber? Sleeping upon the face, or as nearly so as possible, would be the worst position. Certainly upon the back, and without a pillow, would be the single position that would free the septum most from the risk of being covered by sediment. But I have never known but one person who habitually slept in this position. It would be interesting, though by no means conclusive, to learn the position in sleep of glaucomatous patients.

Therapeutics. Not only does this theory of the primary origin of the glaucomatous process seem apriori to meet many or all of the requirements of an explanation, but it also appears to correspond with the clinical demands. All varieties of cases,

the acute, fulminant, subacute, or chronic, would be explainable by it as the natural responses of varying degrees, clearings, and modifications of the sedimental chokings of the filtration-septum.

Both cataract and glaucoma occur in those over forty or fifty years of age, when diminution of secretion and lessening of the general nutritive processes are noteworthy characteristics. Diminished secretion of the dissolving or digestive fluid of the aqueovitreous chamber, even to an infinitesmal degree, in the almost infinitesmal proportions of these areas and functions, would at once produce markedly increased viscidity of the contained liquid—a condition also more liable to occur in connection with the corporeal inactivities of old age, as well as the lessened excretory and vital powers of the tissues generally. When this morbidity is, as it were, chronic, only cataract may arise. When it rises to a certain pitch, clogging, or (as workmen say of sieves and drains) "fouling" of the filtering membrane occurs, and we have the glaucomatous process. This heightened process of glaucoma, too, is prone to occur in the nervous, and worried, and anemic, especially in women, in those generally in whom the expenses of the life-process are in excess of the income—*i. e.*, when there is excess of waste product over excretory and secretory repair—specifically, when the aqueovitreous humor tends to become overloaded with semi-dissolved, non-excreted, viscid matter. (Did a fat person ever have glaucoma?) It may be noted, also, that in glaucoma the lens often become cataractous, whether secondarily from pressure or primarily from morbidity of

the aqueovitreous humor and denutrition remains to be proved. The increased albuminousness and coagulability of the aqueous humor in glaucoma also tally with what the theory would demand. It is exactly as in the physical process of osmosis under pressure outside of the body. The strange clinical fact that the glaucomatous process is often temporarily checked by sleep should not be forgotten. This has a double significance in the light of the theory here proposed.

When we reach the clinical observations that mydriatics intensify the glaucomatous process, and myotics lessen it, we find that hitherto these facts were purely empiric, and the rationale of the action was hidden. But the myotics, eserin and pilocarpin, increase glandular, osmotic, and secretory activity, while mydriatics correspondingly or to a greater degree lessen them. Now, although an increase in the *amount* of the liquid in the aqueovitreous chamber would seem, apriori, to increase the intraocular pressure; it also would lessen the viscidity of the fluid there, and thus lessen the clogging of the filtration-membrane in front. The reason also becomes clear why this action is the more pronounced the earlier in the glaucomatous process it is instituted, and why it is comparatively resultless in late stages, when the anterior filtration-angle and the canal of Schlemm are occluded by pressure and inflammation, and the results of the glandular functions posteriorly are annulled by the same influences.

Nobody has suggested any satisfactory reason for the empiric fact that iridectomy is of use in a cer-

tain class of cases of glaucoma. Sometimes we know it does harm; often it does no good, even in the acute stages, and in the "absolute" or chronic cases, and in glaucoma simplex, it only succeeds in giving any relief in about one-half of the cases. When the tension of the eye is normal it is wholly without effect in reducing tension to subnormality. If the theory I have suggested be proved valid, the explanation of all these facts observed clinically becomes clear, or will become so by investigation. Sclerotomy has undoubtedly effected cure in some cases. But any operative measure, to be effective, must increase the filtration-capacity from the aqueo-vitreous into the aqueous chamber. A simple hole through the suspensory ligament would undoubtedly be absolutely effective—if we could secure this without producing traumatic cataract or luxation of the lens, and when we can safely extract a non-cataractous lens (in conjunction with posterior discission), we may be able to save some remnant of vision in neglected and otherwise hopelessly ruined glaucomatous eyes. In the light of the theory here set forth, iridectomy can only aid in relieving glaucomatous tension by increasing the porosity of the filtering membrane. Just the precise method in which it effects this I leave for future investigation.

Massage, properly and intelligently applied, would seem to be the most clearly indicated therapeutic measure to break up mechanically the clogging process, stimulate additional secretion of diluting and digestive fluid, and arouse normal function generally.

I have had but one case of glaucoma since groping

my way to this theoretic conception of the disease, and that was a typical case, in which vision had been reduced to $\frac{20}{100}$. I began by teaching the patient to some extent the principles, and accurately the manipulations, of massage of the globes of the eyes. There had been no considerable response of the pupil or of the glaucomatous process to eserin. Before proceeding to iridectomy I determined to try massage, and from the day it was instituted improvement began, and the vision steadily rose to $\frac{20}{30}$, with entire cessation of all the symptoms, except that the paralyzed iris still remains somewhat mydriatic and wholly immobile. If the supposed sedimental stasis and choking had not been broken up, stirred up, and helped by means of massage, it would appear that an incision of the sclera beneath the dependent portion of the aqueovitreous chamber, with drainage, and even artificial irrigation of the chamber with a solution of sodium chlorid, would be the natural indication in obstinate and extreme cases.

The foregoing, and especially so far as it relates to cataract and glaucoma, is a study by the method of Zadig, upon a manifestly very small basis of facts. I offer it as a working-hypothesis. I am as well aware as any critic can be of the dangers of extensive inferences from slight data, but all science, in its reachings into the unknown, rests more or less upon Zadig's method, and the only criticism that finally obtains is as to the accuracy of observation of such facts as form the basis of the inquiry, and the inevitableness or firmness of the logical links of the inferential chain. The proof of the whole theory, so far as relates to the last two paragraphs, must

finally rest upon the facts of the existence and running away of the Queen's horse itself, its color, its length of tail, its silver shoes, etc.—that is, in our instance, upon the microscopic study of the parts and processes, and upon the result of the therapeutic tests. These things I must leave to others to carry out. If I have erred, and if the suggestions I have offered are either in part or entirely unjustified, my excuse for the haste of publication, prior to experimental provings, is that I would prefer the just censure of the most unsympathetic critic and disprover, rather than to risk the relief of a single patient by withholding what may turn out to be a new ray of light upon the etiology and cure of a group of formidable diseases.